

NOTES

ACUTE RESPIRATORY DISEASE

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Acute respiratory disorders induced by changes in atmospheric pressure/direct communication between atmosphere, vasculature/pulmonary conditions, diseases (e.g. pulmonary trauma, pneumonia, sepsis, severe burns)
- Impaired alveolar gas exchange → hypoxemia
- Can lead to potentially fatal conditions

SIGNS & SYMPTOMS

- **Hypoxemia:** dyspnea, tachypnea, chest pain

DIAGNOSIS

DIAGNOSTIC IMAGING

- Medical imaging

OTHER DIAGNOSTICS

- Clinical presentation, history
- Arterial blood gases

TREATMENT

OTHER INTERVENTIONS

- Oxygen therapy
- Mechanical ventilation

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

osms.it/ards

PATHOLOGY & CAUSES

- Acute lung condition
- Widespread diffuse inflammation → increased vascular permeability, loss of pulmonary tissue
- Triggered by pulmonary conditions, diseases (e.g. pulmonary trauma, pneumonia, sepsis)

PATHOLOGY

- Refractory hypoxemia, reduced pulmonary compliance, pulmonary edema without

cardiovascular cause (noncardiogenic pulmonary edema)

- **Alveolar barrier cells damaged** → alveolar sacs flooded → impairs air exchange
 - **Pro-inflammatory cytokines released:** tumor necrosis factor (TNF), interleukins
 - Interleukins (IL-1, IL-6, IL-8) → **neutrophil activation** → **toxic substances** (reactive oxygen species) **released** → alveolar and **capillary damage** → oncotic gradient lost → no fluid resorption → fluid in interstitium
- Damaged Type II pneumocytes → surfactant layer malfunction

VISIT...

LANZAROTE
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- Acute inflammatory response → abnormal extravascular fibrin deposition
 - Increased activity of extrinsic coagulation pathway
 - Impaired fibrinolysis

CAUSES

- Systemic infections/septic shock
- Acute lung injury
 - Compromises ability to regulate gas exchange → lungs fill up with fluid in interstitium, alveoli
- Gastric contents aspiration
- Severe pneumonia
- Serious burns
- Mechanical (e.g. near drowning)
- Inflammatory (e.g. pancreatitis)

SIGNS & SYMPTOMS

- Usually begin within first few hours, 1–2 days
- Dyspnea, tachypnea, tachycardia, diaphoresis, low blood oxygenation → cyanosis, diffuse crackles on lung auscultation

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest X-rays

- Bilateral alveolar infiltrate, pulmonary edema with no cardiovascular cause

CT scan

- Bilateral airspace opacities

Ultrasound

- Subpleural consolidations, pleural line irregularities, no lung gliding

LAB RESULTS

- Respiratory alkalosis → respiratory acidosis

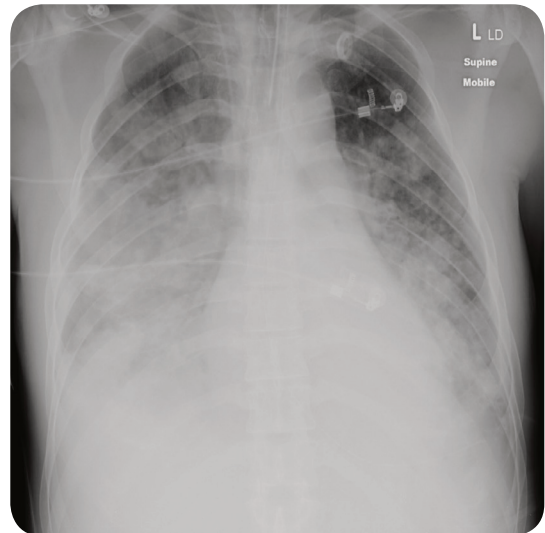


Figure 123.1 A chest radiograph demonstrating diffuse, bilateral, coalescent opacities resembling ground glass.

OTHER DIAGNOSTICS

2012 Berlin definition

- Acute pulmonary injury within week of clinical consultation
- Bilateral opacities on chest X-ray/CT scan unexplained by other pulmonary pathologies (e.g. pleural effusion, lung collapse)
- Respiratory failure without heart failure (noncardiogenic)
- Minimum positive end expiratory pressure (PEEP) of 5cmH₂O
- Reduced oxygen in arteries, reduced partial pressure arterial oxygen/fraction of intake of oxygen ($\text{PaO}_2/\text{FiO}_2$) ratio
 - Mild: 201–300mmHg
 - Moderate: 101–200mmHg
 - Serious: < 100mmHg

TREATMENT

MEDICATIONS

- Antibiotic therapy
 - After microbiological culture, determines appropriate course of antibiotics
- Diuretics
 - Manage fluid output

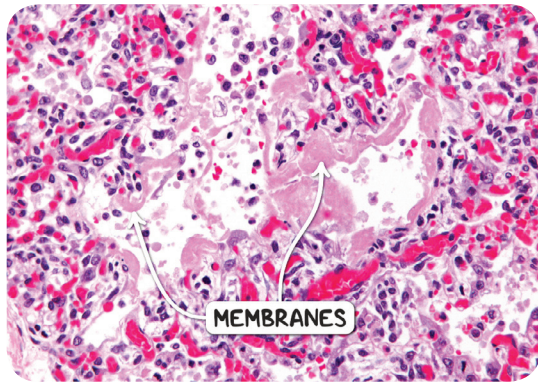


Figure 123.2 The histological appearance of diffuse alveolar damage, the pathological correlate of ARDS. There is a diffuse inflammatory cell infiltrate and pink, hyaline membranes in the alveolar spaces.

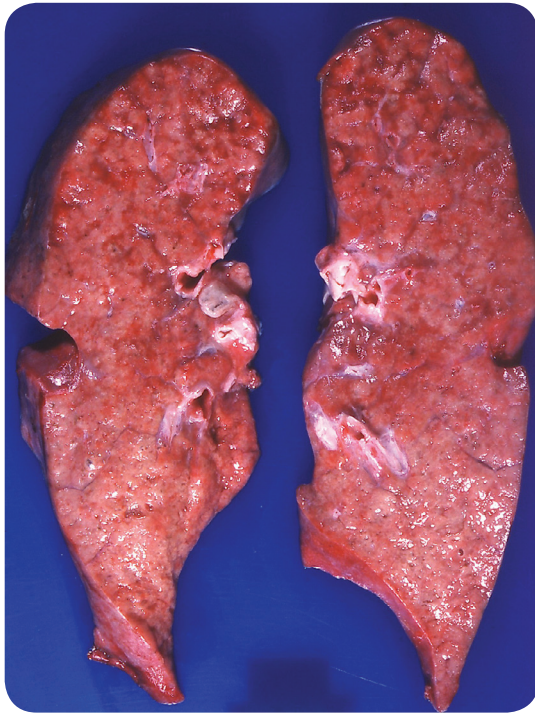


Figure 123.3 The gross pathological appearance of ARDS. There is a diffuse, vaguely nodular infiltrate, most easily visible at the apices.

OTHER INTERVENTIONS

Mechanical ventilation

- Maintain gas exchange to meet metabolic demands
- Endotracheal intubation/tracheostomy (prolonged intubations)
- Monitor parameters
 - **PEEP**: keep alveoli from collapsing, improve oxygenation
 - **Mean airway pressure**: recruit alveoli to open
 - **Plateau pressure**: monitor alveoli for overdistension
- Extracorporeal membrane oxygenation (ECMO)
 - Removes blood from body, artificially removes CO₂, oxygenates red blood cells

ALTITUDE SICKNESS

osms.it/altitude-sickness

PATHOLOGY & CAUSES

- Reaction to exposure to low oxygen concentrations when traveling to high altitude
 - AKA high altitude illness (HAI), acute mountain sickness (AMS)
- Partial pressure of oxygen of inspired air calculated by $PiO_2 \text{ (mmHg)} = FiO_2 \text{ (\%)} \times [Pb \text{ (mmHg)} - 47\text{mmHg}]$
 - FiO_2 : fraction of inspired oxygen, not affected by altitude, remains unchanged in 21%
 - Pb : barometric pressure
 - 47mmHg: vapor pressure of water at 37°C/98.6°F
- In high altitudes, $\downarrow Pb \rightarrow \downarrow PiO_2$
- Partial pressure of alveolar oxygen (PAO_2)
 - Pressure in alveolar space after equilibration with blood
- PAO_2 lower than PiO_2
 - Air enters lungs, humidified by upper airway, partial pressure of water vapor reduces partial pressure of oxygen
 - Continual uptake of oxygen from alveoli by pulmonary capillaries
 - Continual diffusion of CO_2 from capillaries into alveoli
- $\downarrow PiO_2 \rightarrow \downarrow PAO_2, \downarrow PaO_2 \rightarrow$ hypoxemia
- HAI starts at 1.5km/5,000ft, symptoms noticeable above 2.4km/8,000ft

Adaptive mechanisms

- Hypoxemia $\rightarrow$ hyperventilation $\rightarrow \uparrow$ expiration of CO_2 by lungs $\rightarrow \downarrow PCO_2 \rightarrow \uparrow$ pH (respiratory alkalosis)
- $\downarrow PCO_2, \uparrow$ pH inhibit central, peripheral chemoreceptors, decrease ventilation rate
- Within several days $\uparrow HCO_3^-$, $\downarrow H^+$ kidney excretion $\rightarrow \downarrow$ pH $\rightarrow$ stimulation of respiratory center to further increase ventilation
- $\uparrow$ erythropoietin production $\rightarrow \uparrow$ red blood

cell production

- $\uparrow$ 2,3 BPG synthesis $\rightarrow \downarrow$ hemoglobin affinity for $O_2 \rightarrow \uparrow$ release of oxygen to tissues

Measures to avoid HAI

- Acclimatization: ascending slowly to high altitudes, to adjust to decreasing oxygen levels
- Preventative medications: acetazolamide (diuretic); increases bicarbonate kidney excretion

RISK FACTORS

- History of HAI episodes
- Prior exercise/alcohol consumption
- Rapid ascent to high altitude
- Comorbidities that affect breathing (e.g. asthma)

COMPLICATIONS

- Can lead to potentially fatal conditions
 - High altitude cerebral edema (HACE), high altitude pulmonary edema (HAPE)

SIGNS & SYMPTOMS

- Usually appear within 6–12 hours of ascent
- Headache, dizziness, fatigue, nausea, vomiting, loss of appetite, sleep disturbance
- Often improves with time if person does not ascend to higher altitude
- HACE
 - Excessive fatigue, confusion, neurologic deficits (e.g. ataxia, altered mental state)
- HAPE
 - Dry cough, dyspnea

DIAGNOSIS

LAB RESULTS

- Arterial blood gases
 - $\downarrow$ PaO₂, $\uparrow$ PaCO₂, respiratory alkalosis

OTHER DIAGNOSTICS

- Clinical presentation, history of living at low altitude, recent ascent at high altitude

TREATMENT

MEDICATIONS

- Symptom relief
 - E.g. analgesics for headache, antiemetics for nausea
- Carbonic anhydrase inhibitors (e.g. acetazolamide)
 - Increase HCO₃⁻ excretion; treat respiratory alkalosis

OTHER INTERVENTIONS

- Rest
- Descent
- Symptom relief
 - E.g. oxygen to improve breathing
- HACE, HAPE
 - Medical emergencies; require immediate descent/oxygen administration

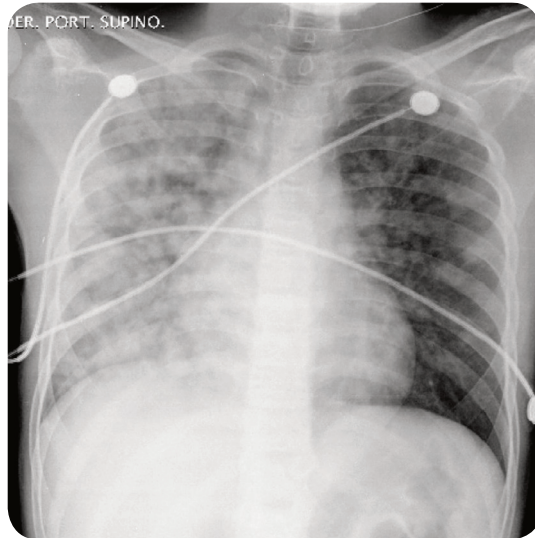


Figure 123.4 A chest radiograph demonstrating acute pulmonary edema in an individual who ascended to 2700m.

DECOMPRESSION SICKNESS (DCS)

osms.it/decompression_sickness

PATHOLOGY & CAUSES

- Gas embolism**, occurs when individuals experience sudden decreases in atmospheric pressure
 - AKA diver's disease
- Air breathed at relatively high pressure (e.g. diver descends from water surface) → inspired gases compressed to higher pressure of surrounding water → $\uparrow$ partial

pressure of oxygen, nitrogen → $\uparrow$ oxygen, nitrogen dissolved in blood, loaded in body tissues

- **Henry's law**: at constant temperature, amount of gas dissolved in liquid directly proportional to partial pressure
- If oxygen, nitrogen quantities high enough → oxygen toxicity/nitrogen narcosis, respectively

- Pressure drops too rapidly (e.g. ascent to water surface) → sum of gas tensions in tissue exceeds ambient pressure → liberation of free gas from tissues due to excess dissolved gases → gas bubbles → vessels blocked, tissues compressed, clotting cascade, inflammation
- Occurs in scuba, deep sea divers, underwater construction workers; during rapid ascent of an unpressurized aircraft
- Caisson disease (chronic decompression sickness)
 - Tunnel workers, moving from caisson to atmospheric pressure

RISK FACTORS

- Right-to-left shunt (e.g. patent foramen ovale/atrial/ventricular septal defect)
- Air travel after diving
- More common in individuals who are biologically male

SIGNS & SYMPTOMS

- Usually develop within one hour of surfacing
- Excessive fatigue, headache
- Depend upon size, location of gas bubbles

Type I DCS

- Skeletal muscles, joints
 - Painful condition, AKA “the bends”; arching of back, posture reminiscent of Grecian bend
- Skin
 - Itching, rash

Type II DCS (more severe)

- Nervous system
 - Paresthesia, amnesia, weakness, paralysis
- Lungs
 - Edema, hemorrhage, atelectasis, emphysema → respiratory distress, AKA “the chokes”; cough, chest pain, dyspnea
- Can progress to permanent injuries/fatal damage

DIAGNOSIS

OTHER DIAGNOSTICS

- Clinical presentation, history of exposure to sudden decreases in atmospheric pressure
- Confirmed if symptoms relieved after recompression

TREATMENT

OTHER INTERVENTIONS

- Hyperbaric oxygen therapy in recompression chamber
 - Under high pressure gas bubbles forced back into solution; slow decompression permits gradual gas elimination via lungs, prevents obstructive bubbles reforming

KEY COMPONENTS OF ACUTE RESPIRATORY DISEASE

	PATHOLOGY/ CAUSES	SIGNS & SYMPTOMS	TREATMENT
ARDS	Inflammation → lung injury → ↑ fluid in alveoli and interstitium → ↑ PAP, ↓ compliance, ↓ gas exchange → hypoxemia	Rapidly progressing respiratory distress: dyspnea, tachypnea, tachycardia, use of accessory respiratory muscles, diaphoresis, cyanosis, respiratory crackles	Mechanical ventilation, fluid management
ALTITUDE SICKNESS	Decreased PiO_2 → hypoxemia Severe cases → HACE, HAPE	Within 6-12 hours of ascent: headache, dizziness, fatigue, nausea, sleep disturbance Severe: excess fatigue, neurological (confusion, deficit), pulmonary (dry cough, dyspnea)	Rest, descent, O_2 , acetazolamide Severe: rapid descent
DECOMPRESSION SICKNESS	Liberation of free gases in tissues; bubbles block vessels, compress tissue, activate clotting, inflammation Etiology: rapid ascent	Type I DCS: muscle/joint pain, itching Type II DCS: neurologic (paresthesia, amnesia, weakness), pulmonary (edema, hemorrhage, atelectasis, emphysema)	Hyperbaric O_2 therapy